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1: J Biochem (Tokyo). 1994 Apr;115(4):648-54. Related Articles, Links

Purification and characterization of a substrate protein for mitochondrial ATP-dependent protease in bovine adrenal cortex.

Watabe S, Kohno H, Kouyama H, Hiroi T, Yago N, Nakazawa T.

Radioisotope Research Institute, St. Marianna University School of Medicine, Kawasaki.

We have purified SP-22, a substrate protein for mitochondrial ATP-dependent protease in bovine adrenal cortex. Native SP-22 showed an M(r) of 350,000 +/- 20,000, and was composed of more than 10 molecules of an M(r) 21,600 subunit. Subcellular and submitochondrial fractionation of adrenocortical tissues revealed that SP-22 was localized in the mitochondrial matrix, suggesting that SP-22 is a natural substrate for ATP-dependent protease, a matrix enzyme. The concentration of SP-22 in adrenocortical mitochondrial fractions was 16 +/- 3 micrograms/mg proteins (mean +/- SD, n = 6) as determined by radioimmunoassay using specific anti-SP-22 antibody. Adrenal cortex showed the highest concentration among the 15 bovine tissues tested, followed by liver, renal cortex, adrenal medulla, heart, and renal medulla. We determined the amino acid sequence of SP-22, which is composed of 195 amino acids. Amino acid 47 was not identified by the sequencer. FAB-mass spectrometry of AA47-AA55 fragment revealed that AA47 was cysteine-sulfinic acid (Cys-SO2H). By a homology search in the NBRF-PIR data base, SP-22 was found to be 91% homologous to murine erythroleukemia cell MER-5 protein, which may have an important role in the induction of differentiation. SP-22 was also homologous to the C22 component of alkyl hydroperoxide reductase in Salmonella typhimurium, thiol-specific antioxidant in Saccharomyces cerevisiae, and some other proteins. Since a segment around AA47 was highly conserved, this residue may be important for the biochemical functions of SP-22.

PMID: 8089078 [PubMed - indexed for MEDLINE]

1: Circulation. 2006 Apr 11;113(14):1779-86. Epub 2006 Apr 3.

Overexpression of mitochondrial peroxiredoxin-3 prevents left ventricular remodeling and failure after myocardial infarction in mice.

Matsushima S, Ide T, Yamato M, Matsusaka H, Hattori F, Ikeuchi M, Kubota T, Sunagawa K, Hasegawa Y, Kurihara T, Oikawa S, Kinugawa S, Tsutsui H.

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BACKGROUND: Mitochondrial oxidative stress and damage play major roles in the development and progression of left ventricular (LV) remodeling and failure after myocardial infarction (MI). We hypothesized that overexpression of the mitochondrial antioxidant, peroxiredoxin-3 (Prx-3), could attenuate this deleterious process. METHODS AND RESULTS: We created MI in 12- to 16-week-old, male Prx-3-transgenic mice (TG+MI, n=37) and nontransgenic wild-type mice (WT+MI, n=39) by ligating the left coronary artery. Prx-3 protein levels were 1.8 times higher in the hearts from TG than WT mice, with no significant changes in other antioxidant enzymes. At 4 weeks after MI, LV thiobarbituric acid-reactive substances in the mitochondria were significantly lower in TG+MI than in WT+MI mice (mean+/-SEM, 1.5+/-0.2 vs 2.2+/-0.2 nmol/mg protein; n=8 each, P<0.05). LV cavity dilatation and dysfunction were attenuated in TG+MI compared with WT+MI mice, with no significant differences in infarct size (56+/-1% vs 55+/-1%; n=6 each, P=NS) and aortic pressure between groups. Mean LV end-diastolic pressures and lung weights in TG+MI mice were also larger than those in WT+sham-operated mice but smaller than those in WT+MI mice. Improvement in LV function in TG+MI mice was accompanied by a decrease in myocyte hypertrophy, interstitial fibrosis, and apoptosis in the noninfarcted LV. Mitochondrial DNA copy number and complex enzyme activities were significantly decreased in WT+MI mice, and this decrease was also ameliorated in TG+MI mice. CONCLUSIONS: Overexpression of Prx-3 inhibited LV remodeling and failure after MI. Therapies designed to interfere with mitochondrial oxidative stress including the antioxidant Prx-3 might be beneficial in preventing cardiac failure.

PMID: 16585391 [PubMed - indexed for MEDLINE]

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L8             0 S L5 (S) L1

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L3	17	"FUruya, Mayumi".in.	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 21:10
S1	187	AOP-1 MER5 PRx3 "peroxiredoxin 3"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 21:05
S2	110664	cardio? cardiac myocyte	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 20:40
S3	5	S1 SAME S2	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 20:47
S4	53899	"chronic heart failure" "heart disease" "cardiovascular disease"	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 20:48
S5	2	S1 SAME S4	US-PGPUB; USPAT; USOCR; EPO; JPO; DERWENT; IBM_TDB	OR	ON	2006/07/18 20:48